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Hostile parenting, parental psychopathology, and depressive symptoms in the offspring: a 32-year follow-up in the Young Finns study

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Abstract

Background: Both hostile parenting and parental psychopathology have been shown to predict depression in the offspring. However, whether and how they interact in predicting the longitudinal course of depression from adolescence to adulthood remains unclear.

Methods: Participants were from the prospective Cardiovascular Risk in Young Finns study, aged 3–18 years at baseline in 1980. We used multilevel modeling for repeated measurements to examine the associations of hostile parenting (i.e., parental intolerance and emotional distance) and parental history of psychopathology with trajectories of depressive symptoms across five study phases from 1992 to 2012.

Results: On average, depressive symptoms decreased in a curvilinear pattern with age. A relatively steep decreasing trend was also observed among offspring of parents with a history of psychopathology but low intolerance. By contrast, among the offspring of parents with a history of psychopathology and high intolerance there was a rising trend in depressive symptoms starting from young adulthood. There was no similar interaction between parental history of psychopathology, emotional distance, and age.

Limitations: Non-standardized, parental self-report scales were used to measure hostile parenting. The observed effects were small, and the depressive symptoms scale applied in the study may not be used for measuring clinical depression.

Conclusions: Parental psychopathology might render individuals sensitive to the unfavorable characteristics of the caregiving environment. Intolerance towards the child can exacerbate the effects of parental psychopathology and have a long-term significance on the developmental trajectory of depressive symptoms over the life-course.

Keywords: parenting; parental psychopathology; depressive symptoms; trajectory

1. Introduction

Affecting approximately 350 million people in all communities, depression is one of the leading causes of disability worldwide (Vos et al., 2012). A substantial body of evidence points to childhood experiences in the pathogenesis of depression (Danese et al., 2009; Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Pirkola et al., 2005), highlighting the role of parenting in the onset of the disorder (McLeod, Weisz, & Wood, 2007). Hostile parenting characterized by emotional distance, neglect, or rejection is a risk factor for the offspring's mental health (Hall, Peden, Rayens, & Beebe, 2004; Oakley-Browne, Joyce, Wells, Bushnell, & Hornblow, 1995; Rojo-Moreno, Livianos-Aldana, Cervera-Martínez, & Dominguez-Carabantes, 1999; Sakado et al., 2000) because it not only undermines self-esteem and promotes maladaptive attitudes (Keltikangas-Järvinen, Kivimäki, & Keskivaara, 2003; Randolph & Dykman, 1998) but also increases neurobiological stress reactivity (Meaney, 2001), which, in turn, contribute to later onset of depression (Sowislo & Orth, 2013; Willner, Scheel-Krüger, & Belzung, 2013). Unfavorable childhood experiences, such as dysfunctional parent-child interactions, thus lay the groundwork for the offspring's future depressive tendencies.

Besides contributing to the development of depression in their offspring through the quality of parenting practices they employ, parents may also pass on vulnerability to the disorder (Rice, Harold, & Thapar, 2002). There is consistent evidence that depression runs in families, as studies have reported parental psychopathology to be associated with up to 13 times higher risk of depression in the offspring (Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005; Weissman, 2006; Wickramaratne & Weissman, 1998). This is likely due to the heritability of depression (Sullivan, Neale, & Kendler, 2000), rendering the offspring of depressed parents inherently susceptible to the disorder. Whereas the heritability estimate of

depression is around 40% in the general population, it reaches staggering proportions (67%) in the direct descendants of clinically depressed individuals (Guffanti et al., 2016).

Whether the association between hostile parenting and depression in the offspring might be dependent on the offspring's susceptibility to depression due to parental psychopathology, however, remains poorly understood. Previous research suggests that there are differences in the way children respond to hostile parenting, depending on the susceptibility of the child (Bradley & Corwyn, 2007; Morris et al., 2002). This susceptibility is suggested to originate from biological differences, making some children more receptive to environmental influences (Belsky & Pluess, 2009). While some evidence suggests that, for example, traumatic childhood events predict depression in the offspring particularly among those who are at risk due to parental psychopathology (Zimmermann et al., 2008), very little research has been conducted to examine the combined effects of parenting and parental psychopathology in the longitudinal course of depression. According to cross-sectional evidence, hostile parenting might be particularly pathogenic in individuals with parental psychopathology (Hammen, Brennan, & Shih, 2004). However, this finding was not replicated in the first, and to date, only longitudinal study examining the effects of parenting and parental psychopathology on the risk of depression in the offspring at a 20-year follow-up (Pilowsky, Wickramaratne, Nomura, & Weissman, 2006). Understanding more about the possible interplay of these factors is necessary for an early detection and prevention of childhood risks for depression in adulthood.

In the present study, we apply repeated measurements to examine whether parental history of psychopathology and hostile parenting interact in predicting the age-related developmental trajectory of depressive symptoms in the offspring from adolescence to adulthood. Although the median age for the onset of mood disorders, such as depression, is around 25-32 years (Kessler et al., 2005), majority of studies examining depression in

individuals with a heightened risk for the disorder have focused on children or adolescents. Consequently, previous research may not have captured the relevant time periods in describing the developmental course of depression in at-risk individuals. It is currently unknown whether and how such risk factors are associated with depressive symptoms in adulthood.

We hypothesized that the association between hostile parenting and depressive symptoms would be more intense in individuals vulnerable to depression, as indicated by parental history of psychopathology. Depression has been shown to share a common genetic background with several other psychiatric conditions, including anxiety, bipolar disorder, schizophrenia, autism spectrum disorder, and attention deficit-hyperactivity disorder (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013; Flint & Kendler, 2014; Guffanti et al., 2016). Specifically, variation in some genes, such as those associated with calcium-channel signaling, is suggested to contribute to general vulnerability to psychopathology. These genes impact brain circuitry involved in a wide range of cognitive and emotional processes that are disrupted in mental illnesses (Bigos et al., 2010). Due to this common biological background of major mental disorders, we used parental history of any psychopathology as a general marker for the offspring's susceptibility to depressive symptoms.

2. Methods

2.1. Participants

Participants were from the ongoing Young Finns study (Raitakari et al., 2008), which is a population-based study following up individuals from six different birth cohorts aged 3-18 years at the baseline in 1980 (*N*=3596). Depressive symptoms were measured for the first time when the youngest cohort had reached the age of 15 (in 1992), and in four

subsequent phases (in 1997, 2001, 2007, and 2012). In the final study phase, the participants were 35-50 years old. The data used in this study are from 2122 participants (59% of the original sample, 967 men and 1155 women) with complete information on hostile parenting, parental history of psychopathology, covariates, and at least one measurement of depressive symptoms. With regard to pattern of missingness in depressive symptoms, 697 (33%) participants had data on depressive symptoms from 5 time points, 445 (21%) from 4 time points, 361 (17%) from 3 time points, 313 (15%) from 2 time points, and 306 (14%) from a single time point (see Supplementary Table 1 for the number of observations by age and measurement year). In this sample, participants were more likely to be women ($p<0.001$) and slightly younger ($p<0.001$), to have higher parental SES ($p<0.001$), to have parents with no history of psychopathology ($p=0.022$), and reported more depressive symptoms in 2001 ($p=0.038$) than those who were excluded. The study was approved by the local ethics committees and conducted in accordance with the Helsinki declaration. Written informed consent was obtained from the participants or their parents, and their treatment complied with APA ethical standards.

2.2. Measures

2.2.1. Hostile parenting. Parenting quality was reported by the participant's parents at baseline in 1980 using a scale derived from Operation Family Study (Makkonen et al., 1981). The scale has two dimensions: parental intolerance and emotional distance towards the child. Parental intolerance of a child's activities was assessed by three items ("In difficult situations, my child is a burden", "I become irritated when being with my child", "My child takes too much of my time"). Emotional distance was measured by four items addressing the emotional significance of the child ("My child is emotionally important to me", "I enjoy spending time with my child", "I am emotionally important to my child", "My child enables me to fulfill myself" [the items were reverse scored]). Responses were given on a 5-point

scale ranging from 1 (totally disagree) to 5 (totally agree). The Cronbach's alphas for intolerance and emotional distance were .68 and .65, respectively. The two dimensions of hostile parenting were examined separately because they are conceptually different and they have been shown to differ in predicting health related outcomes (Ravaja, Katainen, & Keltikangas-Järvinen, 2001). The measures of intolerance and emotional distance share similar elements with the care and indifference subscales of the Parent Bonding Instrument (PBI, Parker, Tupling, & Brown, 1979) , which is one of the most widely studied measures assessing parenting style.

2.2.2. Parental history of psychopathology. Participant's parents' history of psychopathology was assessed in 1980 and 1983 by two indices: the parents' self-reported mental health problems and the parent's use of prescription medicines for psychiatric disorders. The parents were asked: "Have you ever been diagnosed with a mental health problem by a medical doctor?" (yes/no). In addition, the parents were asked to check their prescriptions and list all medications prescribed by a doctor that they were using at the time of the data collection. A parent was coded as "user of psychiatric medication" if he/she reported using any medication targeting specifically depression, psychosis, bipolar disorder, anxiety, or panic disorder. The medications were identified using Pharmaca Fennica 1980, a drug database that is used by medical professionals. Medications that were not prescribed exclusively for the above-mentioned psychiatric disorders but for other non-specific poor mental health conditions, such as alcohol withdrawal delirium or insomnia, were not considered as use of psychiatric medication. A variable for parental history of psychopathology was coded as follows: 0=no parental psychopathology (neither parent reported being diagnosed with a mental health problem nor using any psychiatric medication), 1= parental psychopathology (either parent, or both, had at some point in history been diagnosed with a mental health problem and/or reported using psychiatric medication).

2.2.3. Depressive symptoms. A modified version of the Beck Depression Inventory (Beck & Steer, 1984; Katainen, Räikkönen, Keskivaara, & Keltikangas-Järvinen, 1999) was used to measure self-reported depressive symptoms at five different measurement points. In the modified version, the participants were asked to rate the second mildest statements for the 21 items of the original BDI with a 5-point scale from 1 (totally disagree) to 5 (totally agree). The second mildest statements have been shown to represent the general population variation of the symptoms better than the original scales developed for clinical use (Rosenström et al., 2012). The Cronbach alphas of the scale ranged from .88 to .93.

2.2.4. Covariates. Parental socioeconomic status (SES) was assessed at baseline using two indicators: (1) the mean of mother's and father's years of education and (2) the annual income of the household, both of which were transformed into z-scores (mean=0, standard deviation=1) and then added together. Other measures included participant age (centered at 15 years, which is the age of the youngest cohort at the time of the first depression measurement, and divided by 10), gender, cohort, and parental age at baseline. Gender and cohort were coded as dummy variables.

2.3 Statistical Analyses

There were no interactions between gender and any of the independent variables. The analyses were, therefore, conducted for both genders together. First, we tested for differences in hostile parenting between parents with and without history of psychopathology with Wilcoxon rank-sum test. Next, using five waves of longitudinal data, we applied multilevel modeling for repeated measurements to examine developmental trajectories of depressive symptoms from ages 15 to 50 (i.e., from the earliest age of the youngest cohort to the latest age of the oldest cohort). Examining the data by participant age at the assessment enabled us to take advantage of the age variation by cohort within different measurement points. Each participant contributed one to five person-observations to the data,

depending on the number of phases for which data were available for that participant. Including also those participants who did not have measures of depressive symptoms from all the study phases is justified in multilevel modeling because it corrects for the differences in the mean values of the dependent variable among complete and incomplete cases (Hox, Moerbeek, & van de Schoot, 2010).

We examined the basic characteristics of the developmental course of depressive symptoms first with an unconditional growth model, using only age as the independent variable. We included both linear and quadratic age terms in the model because evidence from previous research suggests that the trajectory of depressive symptoms is curvilinear, declining from adolescence to adulthood and increasing in old age (Sutin et al., 2013). Next, we examined in multivariate models whether participant age, hostile parenting, parental history of psychopathology, and their interactions predicted the development of depressive symptoms. Separate models were built for the two dimensions of hostile parenting. We fitted the models initially with all possible interactions with linear and quadratic age terms and then improved model fit by excluding unnecessary interactions with quadratic age based on the Akaike Information Criterion (AIC). All models were adjusted for gender, cohort, parental SES, and parental age. Finally, we plotted and compared the age-related trajectories of depressive symptoms according to parental history of psychopathology and high (+1 SD above the mean) and low (–1 SD below the mean) levels of hostile parenting. All analyses were conducted using the STATA 13 statistical software (Stata Corporation, College Station, TX).

3. Results

The sample characteristics are shown in **Table 1**. Participants were on average aged 9.63 years (SD=4.84) at baseline. The number (percentage) of participants in birth cohorts aged 3, 6, 9, 12, 15, and 18 at baseline were 408 (19.23%), 394 (18.57%), 409

(19.27%), 384 (18.10%), 309 (14.56%), and 218 (10.27%), respectively. There were 176 (8.29%) participants with a parental history of psychopathology, which is comparable to the rate of diagnosed mental disorders in Finland during the baseline of the study (Aromaa et al., 1989). Among parents using psychiatric medication (n=118), prescriptions targeting anxiety disorder (n=64, 54%) were most common. Several of them had a prescription for depression (n=43, 36%) or psychotic disorder (n=38, 32%), and some parents used medication for multiple conditions, such as for both anxiety and depression (n=8, 7%) or for both psychotic disorder and depression (n=13, 11%). No differences were found in hostile parenting between parents with and without a history of psychopathology (Z=–1.02, p=0.306 and Z=–1.38, p=0.167 for parental intolerance and emotional distance, respectively), suggesting that parental history of psychopathology was not associated with hostile parenting.

Table 1

Bivariate correlations between study variables are presented in **Table 2**. The unconditional growth model with random intercepts and random slopes showed that participants varied (p-values <0.001) in their initial level, i.e. intercept (mean= 2.22, variance=0.24), and their rate of change, i.e. slope of depressive symptoms (mean=–0.13, variance=0.03). On average, depressive symptoms decreased in a curvilinear fashion with age. The intercept and the slope had a negative correlation (r=–0.24), indicating that a higher baseline of depressive symptoms predicted a more rapid decrease in the symptoms with age.

Table 2

Results from the multivariate models predicting depressive symptoms with age, hostile parenting, parental history of psychopathology, and their interactions are shown in **Table 3**. Parental history of psychopathology was the strongest predictor of depressive symptoms in the offspring in both of the models (B from 0.161 to 0.168, p-values<0.01). In addition, both dimensions of hostile parenting predicted higher level of depressive symptoms

(B=0.098, $p<0.001$ and B=0.062, $p=0.007$ for parental intolerance and emotional distance, respectively). The association of parental intolerance with depressive symptoms in the offspring varied with age, and this association was further dependent on parental history of psychopathology (B=0.062, $p=0.031$ for the three-way interaction).

Table 3

Figure 1 presents the age-related trajectories of depressive symptoms for high and low levels of hostile parenting according to the status of parental history of psychopathology (yes/no). Having both a parental history of psychopathology and being also exposed to high parental intolerance predicted the highest level of depressive symptoms. Although, on average, depressive symptoms decreased with age, the trajectory of depressive symptoms showed a different pattern among the offspring of parents with high intolerance. For those who had also a parental history of psychopathology, depressive symptoms decreased until the age of 30, after which they started to rise. Those without parental history of psychopathology showed also a declining, but more long-lasting trend until the age of 40, after which the level of depressive symptoms started to increase. Being exposed to intolerant parenting thus seemed to be particularly pathogenic in participants with a parental history of psychopathology. By contrast, having a parental history of psychopathology and being exposed to more tolerant parenting (i.e., low parental intolerance) was associated with a relatively steep decline in depressive symptoms from adolescence to adulthood. Exposure to tolerant parenting thus appeared to benefit those at-risk for depressive symptoms due to parental psychopathology as they grew older.

Figure 1

4. Discussion

Consistent with previous research (Klein et al., 2005; McLeod et al., 2007; Pilowsky et al., 2006), we found that both hostile parenting and parental history of psychopathology were associated with higher levels of depressive symptoms in the offspring from adolescence to adulthood. Furthermore, hostile parenting characterized by intolerance towards the child seemed to be particularly pathogenic in individuals with a parental history of psychopathology. Although, on average, depressive symptoms decreased with age, the offspring of parents with a history of psychopathology and high intolerance showed a steady increase in depressive symptoms starting from young adulthood. The results suggest that individuals with a parental history of psychopathology might be more sensitive to harmful characteristics of parenting.

Although the association of parental intolerance with depressive symptoms was most intense in individuals having a parental history of psychopathology, parental intolerance was associated with an increase in depressive symptoms in adulthood also among those without parental history of psychopathology. This suggests that intolerant parenting alone is an important risk factor for higher adulthood depressive symptoms in the offspring. While hostile parenting characterized by emotional distance was also associated with a higher level of depressive symptoms, we found no similar rising trend among individuals exposed to emotionally distant parenting. Furthermore, emotional distance and parental history of psychopathology did not interact in predicting the age-related development of depressive symptoms. Whereas we have previously demonstrated the significance of both the parents' intolerance and emotional distance in predicting various negative outcomes in the offspring (Hakulinen et al., 2013; Hintsanen et al., 2010), the results of the current study thus highlight the role of parental intolerance in the development of depressive symptoms. By not accepting the child's normal activity and seeing the child as exhausting and too time-consuming,

intolerant parents may contribute to future depressive symptoms in their offspring. The results are in agreement with previous findings that have shown parental behavior characterized by criticism and disapproval to predict depression-proneness in the offspring more strongly than emotional distance towards the child (Randolph & Dykman, 1998).

With the present data, we cannot determine the reasons why the offspring of parents with a history of psychopathology and high intolerance had a rising trend in depressive symptoms starting from young adulthood rather than already from adolescence. We suggest that although an individual is particularly susceptible to the effects of hostile parenting, some of the adverse effects of parenting on mental health do not necessarily manifest themselves immediately but later in adulthood. It could be that experiencing hostile parenting impacts how individuals experience becoming a parent themselves. During young adulthood, people come across several stressful life-events (e.g., get married, have children, graduate, and start building a career), which could also trigger or exacerbate depressive symptoms. Encountering stressful life-events can afflict particularly those having developed maladaptive attitudes, poor self-esteem, or high reactivity to stress due to parental intolerance. In other words, some of the effects of hostile parenting in individuals susceptible to depression can, and probably do, appear also later in life.

One possible mechanism for the parental transmission of psychopathology is through its negative effects on parenting and the parent-child interaction (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). This, however, was not the case in this study, as we found no differences in parenting depending on parental history of psychopathology. It is also possible that parental psychopathology could have contributed to the higher risk for depression through other factors, such as by generating an unstable home environment. However, parental history of psychopathology was associated with higher levels of depressive symptoms in the offspring even though some of the parents might have only temporarily

suffered from a mental health problem before the offspring was born, suggesting that parental history of psychopathology could reflect biological vulnerability to depressive symptoms in our sample. This kind of biological vulnerability can be based for instance on genetic or neurobiological predispositions, which may increase individual's susceptibility to develop depression (Goodman et al., 2011).

Another potential explanation for our findings involves the accumulation model of childhood risk factors (Evans, Li, & Whipple, 2013), according to which a combination of several risk factors can lead to worse outcomes than exposure to a single risk factor. The accumulation of adversity could explain why individuals with both a parental history of psychopathology and exposure to intolerant parenting had a rising trend and a higher level of depressive symptoms compared to those with only a single risk factor. However, the accumulation model does not explain the relatively steep declining trend in depressive symptoms for those at risk due to parental history of psychopathology but exposed to more tolerant parenting.

The most plausible explanation for our results comes from the diathesis-stress model, according to which predispositional vulnerability interacts with environmental risk factors in the development of psychopathology (Walker, Downey, & Bergman, 1989). The model posits that individual variation in psychiatric disorders depends on whether the inherent vulnerability to psychopathology is combined with an external stressor. Correspondingly, the effect of parental history of psychopathology (i.e., diathesis) was pronounced in individuals who had encountered intolerant parenting (i.e., stress). Besides being a general marker of biological vulnerability to depressive symptoms, parental psychopathology might thus represent a higher biological sensitivity to the unfavorable characteristics of the caregiving environment. Further studies are, however, needed to assess the robustness of these findings. Future studies could include also other factors, such as self-

esteem, to examine mechanisms through which parental psychopathology and hostile parenting are associated with the developmental trajectory of depressive symptoms in the offspring.

The current study has some limitations that should be noted. Individuals in the study sample were slightly different from those who were not included in the study. Although our results may have been affected by selective attrition, attrition rates have been shown to only minimally affect regression estimates when attrition does not depend on the outcome variable (Gustavson, von Soest, Karevold, & Røysamb, 2012). We used non-standardized scales to measure hostile parenting, and the reliabilities of the scales were relatively low. Nevertheless, the scales have shown moderate level of stability over time (Katainen, Räikkönen, & Keltikangas-Järvinen, 1997) and to predict various outcomes in the offspring, such as work stress, temperament, and cardiovascular health (Hintsanen et al., 2010; Josefsson et al., 2013; Keltikangas-Järvinen, 2002). It is also worth noting that parenting quality was mainly reported by mothers. However, maternal reports of parenting quality have been found to be associated for example with mother's role satisfaction and to reflect the general emotional atmosphere in the whole family (Keltikangas-Järvinen, 2002). Because participant age ranged between 3 and 18 years when hostile parenting was assessed, the parenting variable might have had different associations depending on cohort. To explore this, we reran the analyses by standardizing the variables for parenting within each cohort, and found the results essentially unaffected (data not shown). Finally, the observed effects were small, and generalization of our results to clinical setting is limited because the modified depressive symptoms scale may not be used for measuring clinical depression.

The use of prescription psychiatric medication was a sufficient precondition alone for a participant's parent to be categorized as having a history of psychopathology. Consequently, there were also some parents who were classified as having a history of psychopathology

even though they did not report having been diagnosed with a mental health problem. It thus seemed that some parents did not want to report being diagnosed with a mental health problem, although having a diagnosis was evident based on their use of psychiatric medication. We carefully identified medications that were prescribed exclusively for psychiatric disorders during the data collection period and ignored medications that might be prescribed also for other poor mental health conditions. Despite this, we cannot positively rule out the possibility that some of the medications might have been prescribed for other than psychiatric conditions. Nevertheless, the majority (around 80%) of parents using medication reported having also been diagnosed with a mental health problem.

Strengths of the present study include a large study sample and a 32-year follow-up. By applying repeated measurements of depressive symptoms in six different cohorts, we were able to examine the prospective associations of hostile parenting and parental history of psychopathology with the age-related trajectories of depressive symptoms in the offspring stretching from adolescence to adulthood. Unlike most previous research, we used parental reports of parenting quality and offspring reports of depressive symptoms, avoiding some of the problems arising from common method variance. Parental reports of parenting quality are also considered more accurate than the offspring's retrospective reports, which may introduce bias in longitudinal studies on childhood experiences (Hardt & Rutter, 2004).

In conclusion, the present findings suggest that individuals with a parental history of psychopathology might be more sensitive to the unfavorable characteristics of parenting. With regard to the developmental course of depressive symptoms in adulthood, being exposed to parental intolerance can be especially deleterious for individuals with parental psychopathology. For an early prevention of risk factors for depressive symptoms, parents

with a history of psychopathology could be supported to develop and maintain tolerant attitudes towards their offspring.

Conflict of interest

The authors declare no conflict of interests.

Role of funding source

The funding source had no involvement in data collection, designing the study, analyzing or interpreting the results, or reporting the findings.

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Table 1
Descriptive statistics

Variable		N	M or (%)	SD	Range
Offspring characteristics					
Age in 1980		2122	9.63	4.84	3 to 18
Gender (female)		1155	(54.43%)		
Depressive symptoms					
	in 1992	1687	2.13	0.59	1 to 4.57
	in 1997	1483	2.13	0.66	1 to 4.57
	in 2001	1457	2.05	0.67	1 to 4.62
	in 2007	1437	2.04	0.67	1 to 4.67
	in 2012	1216	2.02	0.67	1 to 4.86
Parental characteristics					
SES in 1980		2122	0.22	1.69	-4.13 to 5.69
Age in 1980		2122	38.07	7.38	22 to 64
Parental intolerance		2122	2.08	0.66	1 to 4.33
Emotional distance		2122	1.54	0.49	1 to 4.75
History of psychopathology (yes)[a]		176	(8.29%)		

M = mean; SD = standard deviation. [a] Either parent, or both, having a history of psychopathology.

Table 2
Pearson correlations between the study variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Age	–										
2. Gender[a]	–.04	–									
3. Parental SES	–.12***	.03	–								
4. Parental age	.67***	–.01	–.16***	–							
5. Parental history of psychopathology	.07**	.02[b]	–.13***	.08***	–						
6. Parental intolerance	–.30***	.06**	.09***	–.26***	.03	–					
7. Emotional distance	.10***	.02	.00	.04*	.04	.32***	–				
8. Depressive symptoms in 1992	–.05	–.16***	–.07**	–.04	.07**	.09***	.07**	–			
9. Depressive symptoms in 1997	.02	–.12***	–.06*	–.01	.09***	.07**	.07**	.59***	–		
10. Depressive symptoms in 2001	.03	–.15***	–.07**	.01	.10***	.04	.05	.52***	.65***	–	
11. Depressive symptoms in 2007	.02	–.08**	–.08**	.03	.06*	.05*	.08**	.50***	.55***	.65***	–
12. Depressive symptoms in 2012	.04	–.04	–.07*	.03	.07*	.05	.07**	.48***	.55***	.60***	.71***

N = 1216–2122. Age, gender, and depressive symptoms are offspring characteristics. [a] 0=female, 1=male. [b] Phi coefficient
*$p < 0.05$. **$p < 0.01$. ***$p < 0.001$.

Table 3
Hostile parenting and parental history of psychopathology predicting depressive symptoms in the offspring

	Dimension of hostile parenting			
	Parental intolerance		Emotional distance	
Variable	B	(95% CI)	B	(95% CI)
Age	–0.148***	(–0.196,–0.100)	–0.149***	(–0.196,–0.101)
Age^2	0.027***	(0.013,0.041)	0.025***	(0.012,0.039)
Hostile parenting	0.098***	(0.056,0.140)	0.062***	(0.017,0.107)
Hostile parenting × age	–0.070**	(–0.117,–0.024)	–0.008	(–0.058,0.041)
Hostile parenting × age^2	0.021**	(0.008,0.035)	0.004	(–0.010,0.018)
Parental history of psychopathology	0.161**	(0.044,0.278)	0.168**	(0.053,0.284)
Parental history of psychopathology × age	–0.004	(–0.062,0.055)	–0.004	(–0.062,0.055)
Parental history of psychopathology × hostile parenting	–0.002	(–0.114,0.109)	–0.036	(–0.163,0.090)
Parental history of psychopathology × hostile parenting × age	0.062*	(0.006,0.117)	0.009	(–0.050,0.069)

N = 2122. B = unstandardized beta coefficient; CI = confidence interval. The associations are from two models fitted separately for parental intolerance and emotional distance, adjusting for gender, cohort, parental age, and parental SES. Age was centered at 15 years and divided by 10, and the coefficient reflects how growing ten years older affects the level of depressive symptoms.
*p < 0.05. **p < 0.01. ***p < 0.001.

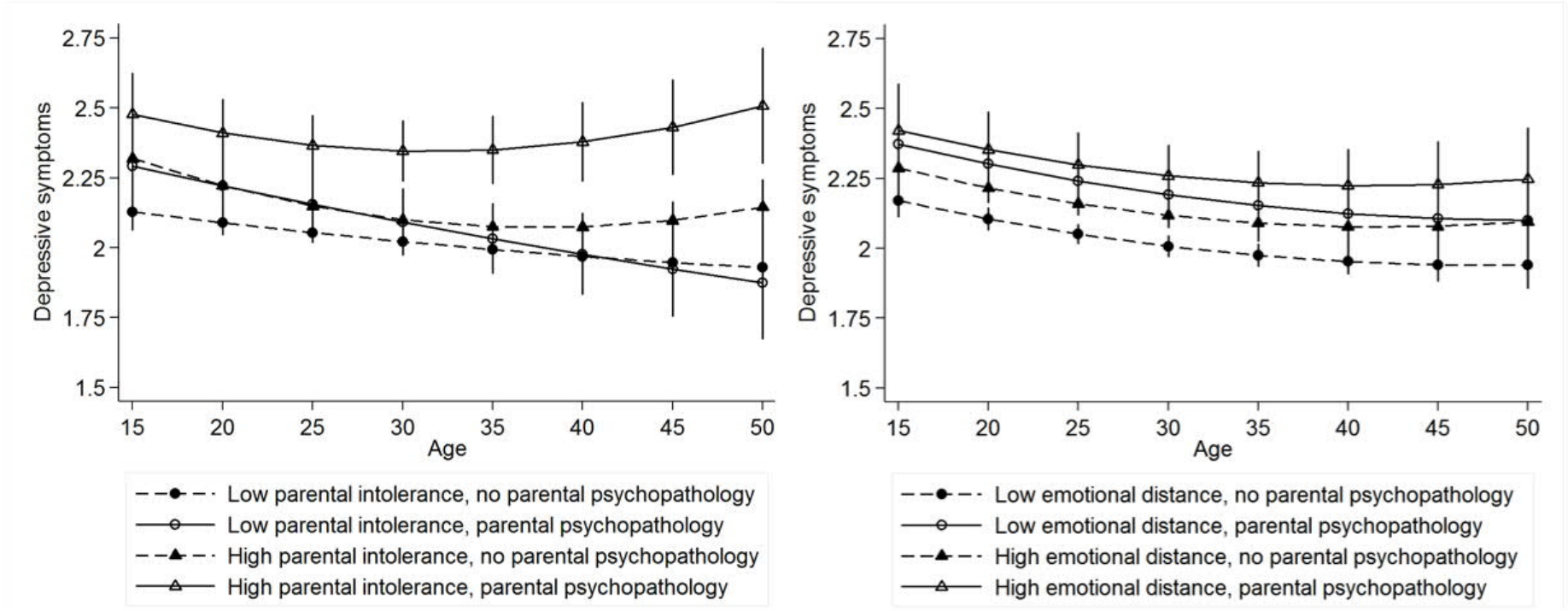

Figure 1. The age-related trajectories of depressive symptoms in the offspring for high and low levels of parental intolerance (left) and emotional distance (right) according to the status of parental history of psychopathology